OBSERVATIONS

ON

H. McFADYEAN.

THE MORBID ANATOMY

OF

SOUTH AFRICAN HORSE-SICKNESS

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THE MORBID ANATOMY
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SOUTH AFRICAN HORSE-SICKNESS.

The three stages of the disease.

The disease termed "Horse-sickness," like all diseases produced by an infective agent, exhibits more than one stage. There is in this disease a definite "stage of incubation" which, extending over a period of from four to eight days, is not attended by any symptoms in the infected animal. The first sign of illness appears at the end of the stage of incubation in the form of an elevation of the temperature; the "stage of fever" thus entered upon developes rapidly, and, having lasted for from three to six days, terminates in the more or less sudden death of the animal. The phenomena of the animal's death constitute in themselves another stage, which although of only a few hours' duration, may provisionally be termed "the stage of death," The approach of death is in almost all cases announced by symptoms which are extremely characteristic, and which are never exhibited before the entrance upon the last stage. The final symptoms are, in the great majority of cases, those of suffocation, and are either gradually or suddenly developed. In some instances the final stage is accompanied by symptoms indicating an interference with the functions of the intestines, but in these cases also some embarrassment of respiration will always have been observed.

The present Report deals with the Pathology of the final stage alone.

There is still much work to be done in the investigation of the various morbid changes which may be found in the body during the stages of incubation and of fever. The observations now presented have been made almost entirely upon the bodies of animals in which the disease has run its complete course and terminated in death, they must be regarded, therefore, as a contribution to our knowledge of the Pathology of "the stage of death" only.

The manner in which the remarkable changes in the body of an animal which has died of the disease are brought about has hitherto remained a mys-

tery. The dropsy of the lungs, the fluid in the pericardial sac, the purple patch in the stomach, and the strange amber-like jelly found among the muscles of the body have so far baffled explanation. It is probable that the cause of these phenomena and of all other changes usually associated in the final stage has now been discovered.

Before entering upon a detailed description of the essential morbid anatomy a summary of the usual changes which may be seen by the unaided eye will be given.

Summary of usual changes found in the body of an animal dead of the disease.

It is as follows:—

White froth around the nostrils.

Thickening of muzzle.

An amber-coloured jelly among the muscles of the neck and in the loose connective tissue of other parts of the body.

A purple, or dark red, patch in the mucous lining of the right, that is the villous, half of the stomach.

An appearance of "congestion" in the mucous membrane of the intestines.

A collection of straw-coloured fluid in the pleural sacs (Hydrothorax).

The pleuræ of the lungs may show rough patches on their surfaces, and hæmorrhages or patches of yellow transudate immediately beneath them.

The lungs will be unusually red, and may show the connective tissue between the lobes to be distended with amber-coloured jelly; the whole organs may be marked by interstitial hæmorrhages, or be loaded with serous fluid.

The connective tissue at the roots of the lungs and around the origins of the great vessels, may be distended with amber-coloured jelly.

A collection of straw-coloured fluid will be found in the sac of the pericardium (Hydro-pericardium).

The surface of the heart may show small hæmorrhages.

The interior of the ventricles, especially the left, may show subserous ecchymoses.

The gums, subconjunctival and subperitoneal tissues may show small ecchymoses.

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The yellow marrow of the thigh bones will be changed in part into a dark red, gelatinous material.

Any or all of these changes may be found in the same body, some of them will, however, usually be found wanting. The most constant, and what may be the termed the "cardinal" *post mortem* signs of Horse-sickness are:—

The changes in the body above enumerated are rarely present together.

The "cardinal" *post mortem* Signs.

The change in the yellow marrow of the thigh bones.

The changes in the lungs.

The patch in the stomach.

The ecchymoses in the left ventricle.

The amber-coloured jelly about the roots of the lungs and among the muscles of the neck and trunk.

The first four of these cardinal signs are almost always found together in the same case. Occasionally one or more are not present,* and if all be absent it is certain that death has not been due to Horse-sickness.

The time which has elapsed between death and the examination of the body has an important bearing upon the appearances found. If, for instance, the body be left lying on its side for some hours, especially if the weather be hot, the fluid in the pleural sac upon the lower side will have a different appearance and density to that in the upper sac; the right lung, right lobe of the liver, and right kidney will appear different to the organs on the left side of the body. The amber-coloured jelly, so characteristic of the disease, may become liquefied by the onset of putrefaction, and from the same cause the cavities of the heart become uniformly stained a deep claret-colour. The importance of not delaying a necropsy cannot be exaggerated. Death is, undoubtedly, sometimes attributed to "congestion" of one lung or to "inflammation" of one kidney when the abnormal appearance of these organs is due entirely to *post mortem* changes.

Importance of early examination of the dead body.

The more important of the naked-eye, *post mortem* appearances of the disease will now be considered in detail.

White Froth around the Nostrils.—In the majority of cases this sign is not present. In about 34 per cent. it appears shortly after death as a rounded mass of white foam attached to the muzzle.

The "Foam-cloud" at the muzzle.

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* The changes in the yellow marrow of the thigh bones may be proved by future observations to be the essential and primary change of the disease.

During the last few hours of life, and while the animal is still on its legs, a viscid, yellowish liquid is often noticed dripping from the nostrils. If this liquid be collected and examined it will be found to be identical in nature with blood-serum, and to have a density slightly greater than that of the fluid which is almost always to be found in the pleural sacs in these cases. When the subject has fallen to the ground and the respiratory movements are coming to a standstill this fluid accumulates in the larger air tubes, and in the nasal cavities and is churned out of the nares in large, glairy bubbles. These, however, do not constitute the "foam-cloud" proper, for this is always formed in the lungs and does not appear at the nostrils until life has been extinct for some little time.

Method of production.

To understand how the foam-cloud is produced, we must remember that the tissue of the lungs is highly elastic, and that in these cases of the disease the elastic lung tissue is tensely distended with serous fluid. After death the tense, œdematous lung gradually contracts, and this contraction expels some of the fluid from its interior in which situation are many alveoli and bronchioles still filled with air. The mingled fluid and air thus passively expelled from the lungs constitute the foam-cloud, which, creeping up the trachea, appears at the nostrils. The *post mortem* accumulation of gas in the stomach and intestines, by causing pressure on the diaphragm, assists in expelling the froth which has collected in the air passages.

An interesting demonstration of the method by which the foam-cloud is produced is sometimes seen when the lungs from a case marked by pulmonary œdema are rapidly removed after death. Upon cutting such a lung right through a white froth will be seen to gradually exude from the cut ends of the larger air tubes, and if the divided lung be left undisturbed the escaping froth will accumulate in rounded masses of foam-cloud which may attain a considerable size.

The changes with time.

If allowed to remain for some hours, the foam-cloud at the muzzle will entirely disappear, collapsing into a small pool of serum. It has been seen, however, but little changed at the end of 24 hours.

Significance of the sign.

The viscid fluid dripping from the patient's nostrils, and the foam-cloud at the muzzle of the cadaver have both the same nature, origin and significance. The material is serous fluid and is derived from the lungs. Whenever the fluid or the foam is seen we may expect to find the lungs œdematous. This is

not, however, the whole of the matter, for we shall almost always find, in addition to the œdema, a large collection of fluid in the pleural sacs. Out of fifteen instances† in which serum flowed from the nostrils or the foam-cloud formed, excessive dropsy of the pleural sacs was found in twelve; in two only of the fifteen cases referred to was excessive pulmonary œdema found without pleural dropsy. In the remaining case excessive œdema and excessive dropsy coexisted.*

In the great majority of cases then, a dripping of serous fluid from the nostrils during life indicates the existence of hydrothorax. If the sign be combined with that next to be described, the existence of pleural dropsy amounts nearly to a certainty.

Coarseness of muzzle.

THICKENING, OR COARSENESS, OF MUZZLE.—As often as not this change is absent; it appears to be present in about 50 per cent. of cases.

Morbid anatomy.

If the thickened lips be cut into, the connective tissue will be found to be abnormally succulent. Rarely, but certainly occasionally, recent extravasations of blood will be found beneath the mucous surfaces.

In some instances, in addition to the thickened muzzle, a soft tumefaction of the eyelids will be found, which is due to an extension of the same process. The designation "Dikkop form" for such cases of the disease is not free from objection, and if employed at all should be used in a strictly clinical sense only. As will be shown at the end of this report all the changes which characterise fatal Horse-sickness are due to one and the same cause.

The method of production.

Œdema of the muzzle is not the result of an embarrassment of the whole circulation, for under such circumstances lax tissues in other parts of the body, especially those which are dependent, such as the scrotum and the mammillary nipples, would also be affected. The change appears to be due to an impediment to the flow of blood from the head and neck. This impediment is brought about in nearly all cases by pressure upon the Anterior Vena Cava in the chest. This pressure is the result of fluid collections in the pleural and pericardial sacs. A further cause of impeded blood-return is found in some cases, and in such tends to increase the muzzle-œdema, this is pressure upon the Jugular Veins by transudate among the neck muscles.

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* For a definition of "excessive" dropsy and œdema see pp. 9 and 11.

† Only 12 of these cases are included in the table appended to this report.

Out of nineteen bodies which showed the sign, nine proved to have excessive pleural dropsy and ten excessive pericardial dropsy.* No case showing the change has been examined in which one or other of these causes of intra-thoracic pressure was not present. Unlike the swelling of the temporal fossæ the œdema of the ~~muscles~~ muzzle does not seem to alter to any extent after death.

It is worthy of note that in each of nine instances in which muzzle-œdema existed in association with the escape of fluid or the foam-cloud from the nostrils, an excessive dropsy of the pleural sacs was present.†

Bulging above the eyes.

Elastic Œdema of the Temporal Fossæ (Swelling of the hollows above the eyes).—An elastic swelling in the temporal fossæ is found in a little more than half the cases. This sign varies greatly both with the age of the subject and the time since death. It may increase in size or appear for the first time after death, while on the other hand it may diminish or entirely disappear after that event.

If, in a well-marked case, the temporal fossæ be dissected immediately, after death, a turgescence of the deeply-placed temporal veins will usually be the only abnormality discovered. Sometimes a small quantity of serous fluid or yellow gelatinous material may be found below the pad of elastic fat which naturally occupies this hollow, the amount of the transudate is not, however, in any way commensurate with the extent of the bulging of the skin. The swelling of the temporal fossæ cannot therefore be considered an ordinary œdema,

Cause

but may be looked upon as an indication of an engorgement of the deep plexus of veins in this region. The gradual escape of blood from the deep temporal plexus into the larger veins may explain the diminution of the swelling which may occur after death. The phenomenon of the increase of the swelling after death is explained by the driving of blood into the plexus owing to the putrefactive distension of the abdomen.

In 16 out of 20 cases‡ in which swelling of the temporal hollows was noted, a gelatinous transudate was found on either side of the windpipe in close relation to the jugular veins. This gelatinous material in the jugular channels,

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*For a definition of "excessive" pericardial dropsy see p. 14. Only 14 of the 19 cases referred to are included in the appended table.

†The concurrence during life of the two signs, coarseness of lips and dripping of serum from the nostrils, might well be taken as an indication for immediately tapping one or both pleural cavities.

‡Fourteen only of these are included in the synoptic table appended.

by the pressure which it exercises upon the veins, appears to be the main factor in the production of ante-mortem swelling of the temporal fossae.

Amber-coloured Jelly in the jugular channels and other connective tissue spaces of the body. This *post mortem* appearance is very characteristic of the disease. It is not, however, an essential phenomenon, for no trace of it may be found in an undoubted case, and it also occurs in small quantities in horses dead of other diseases. Out of 34 bodies examined, an intermuscular collection of jelly was found in 17, and it may be taken that this change is present in about 50 per cent. of cases. Amber Jelly,

The substance in question presents itself as a clear, light amber-coloured, lobulated and gelatinous material. Its specific gravity is about 1,020, but, like all the vascular transudates in this disease, the density varies in different cases. Microscopically examined the substance proves to consist of minute fibrils embedded in a structureless jelly contained in the meshes of loose connective tissue. Chemically the composition is found to be approximately the same as that of blood serum. The nature of the jelly is obviously that of a coagulated transudate from the blood vessels. Nature and Origin.

During life the amber jelly is not present as such, it exists in the form of a liquid. If the body be opened immediately after death the positions in which the jelly is to be looked for will be found occupied by a clear, straw-coloured fluid, and if this fluid be watched it may be seen to become a solid jelly in a few minutes. After some hours, whether in or out of the body, and with especial rapidity in hot ~~water~~ weather, this jelly will again become a liquid.

The most common situation for "amber jelly," as, for the sake of brevity, this material will now be called, is in the jugular canals on either side of the trachea. In this situation it is found to form a sort of matrix in which the vein, the carotid artery and the vagus nerve appear to be embedded. From the jugular canal it is often found continued backwards into the anterior mediastinum in relation with the trachea which it accompanies to its bifurcation. Occasionally the jelly is found at the base and in the middle of the neck without any in the thorax. Nearly, if not quite, as common a situation for amber jelly is in the subperitoneal connective tissue of the ventral abdominal wall. Other likely situations are—in the connective tissue planes between the oblique abdominal muscles, between the subscapularis and serratus mag- Usual situations of amber jelly.

nus muscles, among the muscles of the back, chest, shoulders and haunches, surrounding the larger arteries in the extremities and beneath the peritoneum of the pelvic and dorsal abdominal walls. It may be remarked that the distribution of this transudate is not that of a general œdema; the nature of the substance is, moreover, identical with the jelly found beneath the pulmonary pleura, and in the connective tissue of the lungs.

Amber jelly a local œdema of unusual nature.

The presence of this amber jelly may be considered to indicate an œdema of a somewhat unusual variety, the peculiarities of which lie in its restricted distribution, the rapidity of its formation and the property which the transuded fluid has of coagulating after death. The explanation of these peculiarities will be referred to at the end of this report.

Hydrothorax.

Fluid in the Pleural Sacs.—The amount of fluid occurring naturally in the pleural sacs of the horse may be put down at from 0 to 3 or 4 cc.; any quantity exceeding 20 cc. is certainly abnormal.

A collection of fluid in both pleural cavities is one of the most constant signs of death from Horse-sickness; out of 35 bodies examined it was found in 34 instances, i.e., in 97 per cent.

Never one-sided but usually unequal on two sides.

It has been especially noted that the fluid has never been found confined entirely to one pleural sac. The quantity on the two sides is, however, rarely equal; sometimes there is more on the right, sometimes on the left.

Out of 32 instances in which accurate measurements were made 16 showed an excess in the right sac, 10 in the left, and 6 an equal quantity on the two sides. The difference in the quantities on the two sides is often very marked, for example—case "51" showed 800 cc. on the left, and 3,500 on the right side; "27" proved to have 200 cc. on the left and 100 cc. on the right.

The animal usually dies lying upon that side which contains the larger quantity of fluid.*

The average quantity of total pleural transudate in the 34 instances first referred to, was 1,869 cc. In 10 cases it exceeded 3,000 cc., in 7 it fell between 1,000 and 3,000 cc., and in 15 between 100 and 1,000 cc. The greatest quantity found was 6,000 cc., and the least 20 cc. In estimating the amout of this

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* The tongue is usually found lolling out of the mouth after death, the side of the mouth from which the tongue protrudes affords a simple indication of the side on which the animal died.

fluid the contents of both pleural sacs have been added together; a total exceeding 3,000 cc. will be termed an "excessive" pleural dropsy.

The appearance of the pleural fluid varies somewhat, the typical specimen is clear and of a light straw-colour. Some turbidity is often noticed if the examination be made immediately after death. Occasionally the colour in a recent body is a redish-amber; as a rule, however, any redness of tint is to be taken as evidence that death has taken place some hours previously. If the necropsy be delayed for from six to eighteen hours the pleural fluid will, as a rule, be found still clear, but of a cherry-red or claret colour. Twenty-four hours after death the fluid has assumed a dark red tint. These changes in colour are one of the first effects of approaching decomposition, and are due to an escape of colouring matter from the blood.

Appearance of the fluid.

Changes with time.

There has not been any evidence to suggest that alteration in the bulk of the pleural dropsy, either an increase or decrease, takes place after death. Its formation is an essentially vital process, and is dependent upon a still active circulation.

A difference is often to be observed in the appearance, as well as in the density of the fluids in either pleural sac. These differences are developed *post mortem,* and depend upon which side the body has lain. The fluid in the lower sac is darker and slightly more dense than that in the upper.

Differences of the two sides.

The specific gravity of the pleural transudate has been found to vary between 1,012 and 1,032. The usual density may be stated to be 1.018, and it has been found that variations from this figure are principally due to differences in time between death and examination. In no instance in which the examination was undertaken within four hours of death did the density of the fluid exceed 1,020. In all the cases in which a fluid was found, the specific gravity of which exceeded 1,020, the necropsy had been delayed for longer than six hours. Unlike the "amber jelly" transudate of Horse-sickness the pleural fluid has not, in any case, been found to coagulate either before or after removal from the body. Its reaction, when fresh, is faintly alkaline, and its chemical composition appears identical with that of blood serum.

Density of the fluid.

Nature.

As bearing upon the causation of this dropsy of the pleural sacs the following observations have been made. In each of 31 cases the parietal pleura, excluding that which covers the diaphragm, was found of normal appearance.

Considerations which suggest the method of production.

In 25 out of the 31 cases the pleura which covers the lung was found to be abnormal: in 6 only was it normal. Of the 25 showing abnormality of the visceral pleura 13 exhibited areas, of greater or less extent, of gelatinous transudate beneath the serous membrane; in 7 there were subserous ecchymoses, and in 5 subserous ecchymoses and gelatinous transudate occurred in combination.

The relation between the different conditions of the visceral pleura, and the amount of the dropsy is instructive. In the 13 cases exhibiting subpleural amber jelly, the average amount of fluid in the sacs was 3,414 cc.; in those 5 with subpleural amber jelly and ecchymoses in combination, the average amount was 1,317 cc.; in the 6 with an apparently normal membrane the average amount was 688 cc., whilst in the 7 with subpleural ecchymoses alone the average was 541 cc.

It appears, therefore, that the cause of excessive dropsy of the pleural sacs is associated with that producing patches of amber jelly beneath the visceral pleura. This suggestion gains still greater force when we find that in each and all of the 10 cases which showed excessive dropsy (i.e., a total exceeding 3,000 cc.) patches of subpleural jelly were also present. When the nature of the processes responsible for these two morbid changes comes to be discussed, it will be readily seen that a pleural dropsy might certainly occur apart from any obvious affection of the pleura, but that the converse of this would be a pathological impossibility.

Lungs and their pleuræ

THE CONDITION OF THE LUNGS AND THEIR PLEURAL COVERING.—If the theory put forward at the end of these observations be the correct explanation of the cause of death in Horse-sickness, it necessarily follows that *the lungs are always affected* to a greater or less extent. That they are, as a rule, obviously and grossly affected is well known, instances, however, occasionally occur in which the morbid changes are not apparent to the untrained eye. In these cases an undue redness of the lung tissue* is always present, and is due to a change which, had the animal lived for a few hours longer, would have been followed by all the familiar morbid signs of the typical case.

As already suggested, the accumulation of fluid in the pleural

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* This redness is often sufficiently marked to justify one in describing the cut surface of the organ as being of a scarlet colour. The natural hue of the lung tissue is a French grey or dirty pink.

cavities may be considered an indication of some abnormality in the lung or its serous covering. In a few instances of excessive pleural dropsy, the lung, with the exception of a patch or two of subpleural jelly, and some undue redness, has appeared to the unaided eye to be almost normal. There can, however, be no doubt that in such cases, as already stated, the lung is the arena in which the first act only of the usual pathological tragedy has been played—the second act has only just commenced.

The changes in the lung which are typical of the disease, are as follows:—

The weight is increased.

The pulmonary tissue proper is unusually red, and may show numerous small areas of recent haemorrhage.

The connective tissue of the whole organ is œdematous. It is to this œdema that the increase in weight is principally due. When the weight of both lungs exceeds 11·34 kilogrammes an "excessive" œdema is said to occur. In some parts of the lung the air vesicles and smaller air tubes have become flooded with a serous fluid. When such a part is cut through a frothy liquid streams from the cut surface. Occupying the connective tissue partitions between the lobes and lobules, planes of amber jelly will be found (in about 33 per cent. of cases). These deposits become thicker and more numerous towards the surface of the organ. At the surface, the interlobular deposits become continuous with similar deposits beneath the pleura. This usually-coagulated transudate has been discovered in a liquid condition, and seen to gelatinise while being collected in a vessel for examination.

The loose connective tissue surrounding the bronchi and pulmonary end of the trachea is found (in 33 per cent. of cases), distended with amber jelly. This jelly may be so abundant as to envelope the bronchial glands. It may often be traced, in diminishing quantities, accompanying the air tubes into the depths of the lung.*

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* The amber jelly in the connective tissue partitions of the lung may, therefore, be stated, for descriptive purposes only, to originate in two situations and spread thence into the organ; these situations are:—(1) The subpleural tissue: (2) the peribronchial tissue.

The bronchial lymphatic glands may be unduly juicy, and may exhibit points of hæmorrhage on section.

The pleural covering of the lung, especially on the dorsal surface, may appear thick and opaque. The change is due to an œdema of the fibrous basis of the membrane.

Beneath the pleura appear areas, varying in extent and thickness, of clear amber jelly. This material may, however, be only partially coagulated or quite liquid. The serous membrane covering these patches of transudate will often be found to strip off with the greatest ease. The surface of the patches of subpleural amber jelly is frequently opaque, of whitish colour, nodulated, rough, slightly shaggy and marked by small, bright red ecchymoses.

The pleura may be marked by recent ecchymoses of varying size. These hæmorrhages are either confined to the substance of the membrane or extend some little way into the subjacent tissue.

The trachea may carry a cushion of amber jelly between its fibrous wall and its mucous lining in that part of its circumference where the cartilaginous rings are incomplete.

The bronchioles, bronchi and trachea are frequently filled with a white froth. This froth is identical in nature with that which, as previously described, may form the "foam-cloud" at the nostrils after death.

The extent to which the lungs show morbid changes varies greatly.

One, all, or any combination of these various changes may be found in the same specimen.

The commonest situation for tracts of subpleural amber jelly is along the thin, free edge of the ventral margins, and on the dorsal and diaphragmatic surfaces of the lungs. A common region for an extensive deposit of amber jelly is between the inner lobe of the right lung and the opposed surface of the left lung. The most frequent situation for ecchymoses and shaggy patches is upon the dorsal surface of either organ near its diaphragmatic border.

Alterations with time.

Modifications of the appearances in the lungs, consequent upon delay in commencing the examination, may be briefly stated to be—a tendency to liquefaction of the gelatinous deposits and more or less obscuration of petechiæ and

similar changes by *post mortem* staining. In a late necropsy the lung on the lower side will always appear darker and more engorged than that on the upper side.

In no case marked by excessive hydrothorax has a collapse of the lung been found. This fact is of particular interest, and is further evidence that the pleural transudate is derived, not from the whole inner surface of the pleural sac, as in an ordinary dropsy, but from the lung itself. It is apparent that the pathological processes responsible for the lung-changes primarily affect the vascular system. The pulmonary system is not, however, alone involved. Œdema of, and hæmorrhages into, the bronchial glands; the presence of coagulated transudate around the bronchi, in the wall of the trachea, in the connective tissue partitions and beneath the pleural covering of the lung, may all be taken as evidence that the bronchial circulation is implicated as well as the pulmonary. The reason why both these vascular systems are affected will be suggested later.

The vascular system responsible for the lesions in the lungs. Both pulmonary and bronchial systems involved.

It is especially noteworthy that evidences of an inflammatory process in the lungs are conspicuously absent. There cannot be found any traces of those more solid products of inflammation which accumulate in the hepatised lung of a croupous pneumonia. The air cells and smaller tubes may be filled with fluid, but this fluid is of the nature of a serous transudate; it in no way resembles the muco-purulent exudate of a broncho-pneumonia.

Absence of changes due to inflammation.

Fluid in the Pericardial Sac.—The pericardial sac does not naturally contain any fluid, its lining membrane is merely kept moist. An accumulation of fluid in this serous cavity has been found in 34 out of 35 cases examined, i.e., in 97 per cent.

Hydropericardium.

In colour, the pericardial transudate varies from a very pale straw tint to a dark maroon; whatever the colour may be the liquid is nearly always quite clear. If the examination be made within a few hours of death the colour is some shade of yellow, lighter or deeper according to promptness or delay in undertaking the necropsy. Eighteen or twenty hours after death the fluid usually appears of the colour of port wine. The density varies from 1,014 to 1,026. It is a general rule that the density of the fluid rises if the body remain unopened for some hours. Out of 17 necropsies made within 10 hours of death there was no single instance in which the specific gravity reached

Character of fluid and changes with time.

1,020; on the other hand out of 9 examinations made between 15 and 22 hours of death the specific gravity of this fluid exceeded 1,020 in 5 instances. It will be observed that the post mortem changes in colour and specific gravity of the pericardial effusion are of a similar nature to those which the fluid in the pleural sacs undergoes. By attention to such details as these it would appear possible, if circumstances called for it, to make a fairly accurate estimate of the hour at which death took place.

The pericardial effusion is of alkaline reaction, and has a chemical composition identical with that of blood serum. Unlike the pleural transudate it has been occasionally found to coagulate after removal from the body.

The average amount of liquid present in the pericardial sac, in 34 cases, was 414 cc. In 3 it exceeded 1,000 cc.; in 11 it fell between 500 and 1,000 cc.; in 12 between 100 and 500 cc.; and in 8 it measured less than 100 cc. . The largest amount found was 1,380 cc., and the least 10 cc. A quantity measuring 500 cc. or more has been considered as an "excessive" dropsy of the sac.

Fluid collections in Horse-sickness transudates, not exudates.

The characteristics of the fluids which accumulate in the pleural and pericardial sacs in Horse-sickness are essentially those of *mechanical transudates* rather than of inflammatory exudates. Their low density, freedom from leucocytes and the rarity with which they coagulate, all favour this view. More convincing, however, than the peculiarities of the fluids themselves, is the character of the changes in the serous membrane from which they escape. The parietal layer of the pleura and pericardium has not, in any case, appeared abnormal, the visceral layer, that which covers the lungs or heart, has frequently showed morbid changes.* It is, therefore, a reasonable supposition that the transudate escapes into the sac from the visceral rather than from the parietal layer. The essential changes in the visceral pleura and pericardium, which will be discussed later, cannot be considered as phenomena of inflammation.

In a general way the amount of pericardial transudate was found to be inverse to the amount of pleural transudate, where the one was in excess, the

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* For the abnormalities of the visceral pleura see p. 12, and for those of the visceral pericardium see p. 16.

other, as a rule, proved deficient.* The reason for this peculiarity will be explained to some extent when the details of the methods by which the fatal transudates arise come to be discussed.

Probable cause pericardial transudate.

The immediate cause of the pericardial transudate is probably an obstruction to the circulation of the blood in the vessels which supply the tissues of the heart. In some cases the flow of dropsical fluid from the surface of the organ is the only indication of this obstruction; usually, however, there are signs beneath the serous membrane lining the cavities, and often beneath that which covers the surface, which give further demonstration of a damaged blood supply. On the inner surface of the heart, subserous ecchymoses appear, and on the exterior, ecchymoses or tracts of amber jelly. That these abnormalities of the epi- and endo-cardium are usually associated with, and have, therefore, in all probability, the same cause as the pericardial dropsy, is evident from the following considerations:—

Out of the 34 cases examined the transudate exceeded the average of 414 cc. in 14 instances and fell below the average in 20 instances. Of the 14 which exceeded the average, the epicardium was normal in 35 per cent. only (5 cases), and the endocardium in 7 per cent. (1 case). Of the 20 which fell below the

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*The general inverse ratio between the quantities of pericardial and pleural transudates is shown in the following table:—

	Number of cases.	Average amount of pericardial transudate.	Average amount of pleural transudate in same cases.
Cases in which pericardial transudate exceeded 1,000cc. ...	3	1,260cc.	490cc.
Cases in which pericardial transudate measured between 500 and 1,000 cc....	11	678cc.	1,471cc.
Cases in which pericardial transudate measured between 100 and 500cc.	12	208cc.	2,576cc.
Cases in which pericardial transudate measured less than 100cc.	8	43cc.	2,094cc.
All cases examined	34	414cc.	1,923cc.

average the epicardium was normal in 75 per cent. (15 cases), and the endocardium in 10 per cent. (2 cases).

The Heart.

The Condition of the Heart, Pericardium and the Origins of the Great Vessels.—It is rarely that these organs are quite normal, in only one instance out of 34 was no morbid change discovered.

The pericardium has always presented a smooth, glossy, serous surface. The fibrous layer of the sac towards its attachment around the great vessels may be infiltrated with a yellow liquid (1 case), or with a bulky, yellow coagulum (2 cases.)

The surface of the heart (or epicardium) may show recent petechiæ or larger ecchymoses (3 cases). The connective tissue of the grooves in which the coronary vessels run, may be distended with amber jelly (4 cases). These two signs, petechiæ and jelly, may appear in conjunction (5 cases).

Amber jelly may be discovered deeply embedded at the base of the heart around the origins of the Pulmonary Artery, Aorta and Pulmonary Veins (9 cases); or deeply infiltrating the septum between the auricles and that between the auricles and ventricles (1 case).

The bases of the cusps of the tricuspid and mitral valves may be found distended with amber jelly and showing small petechiæ (2 cases).

The endocardium presnts subserous ecchymoses in about 90 per cent. of cases. Out of 27 instances in which the distribution of these subserous hæmorrhages was recorded, 16 showed the sign in the left ventricle only, 10 in both ventricles—nearly always more developed in the left—and 1 in both ventricles together with the left auricle. The most constant location for the ecchymosis, and that in which, when present, it is most developed, is at the base of the musculus papillaris, springing from the septal wall of the left ventricle.

The surface of the Pulmonary Artery at its root showed numerous minute petechiæ in one case.

Influence of time.

The appearances within the cavities of the heart do not alter until the onset of putrefaction. In one case the heart was removed immediately after death, opened, and the condition noted. The organ was then replaced in the body and examined again in twelve hours time; no change in the appearances had taken place.

Post mortem staining of the endocardium was on no occasion noted before 20 hours after death. In mentioning this fact, it may be remarked that death

from Horse-sickness, unlike that from septicæmic diseases, is not followed by an unduly early decomposition.

The changes in the heart are obviously not inflammatory in nature: there is no dulling of, or exudation of lymph upon, the surface of the serous epicardium: no purulent infiltration of the myocardium and no ulcerative destruction of the endocardium. The apearances are such as would follow an interference with the special blood-supply of the organ and its appendages. The nature of the changes is evidently identical with that of those occurring in the lungs and other parts.

Absence of signs of inflammatory processes

The Change in the Stomach.—A change, exceedingly characteristic, in fact pathognomonic, of the disease, is usually found in the mucous membrane of the stomach. The frequency with which the villous half of the stomach presented this abnormality was observed some years ago by the Director of the Laboratory.

The right half of the stomach.

Out of 35 cases, the stomach appeared as follows:—Normal in 3; showing a very faint mottling in 3; showing a lightly-tinted, mottled patch in 12; showing a typical "port wine patch" in 11, and a very pronounced degree of the same change in 6. Excluding those instances in which the change is noted as being very slight, it is found, therefore, that the villous stomach presents obvious change in about 83 per cent. of cases. The typical sign shows itself as a more or less circular area, clearly defined from the surrounding normal surface, having a diameter of about 5 inches, and a reddish-purple colour. The patch is situated on the side of the greater curvature near to or touching the junction of the villous and cuticular regions. Occasionally the villous mucous membrane is affected throughout its entire extent. The surface of the area is usually covered with a thin, semi-transparent membrane which has the property of "rucking up" when the finger is lightly drawn across it. Sometimes this easily-detached membrane, which consists of a few cells embedded in a tenacious mucus, is found to have adhered to the food, and to have been emptied out of the stomach. In addition to this false-membrane, particles of food are usually found attached to the affected surface.

If the patch be cut right through, the subjacent areolar, muscular and serous coats will appear quite normal. The "port wine patch" is entirely confined to the glandular layer of the stomach wall.

The abnormality in the stomach confined to mucous and submucous layers.

Degrees of the change.

As already stated, there are many degrees of intensity in this sign; the change varies from a mere mottling of a pale lavender tint, to a clearly defined area of a deep, red, mulberry colour. When fully developed the patch may appear nearly black, and in such cases there is usually some loss of surface in the form of irregularly-circular depressions. There may also, in these extreme cases, be a slight escape of blood from the surface, and this will usually be found to have stained the mass of food contained within the viscus.

Influence of time.

The only change in the stomach-appearances which results from a delayed examination, is that the false-membrane which overlies the affected region becomes more obvious, and will then be found rucked up in prominent ridges. The appearance of the subjacent mucous membrane remains unchanged for many hours.

Beyond the presence of this patch of lividity, or, as it may be more descriptively styled, "the port wine patch," the villous mucous membrane rarely presents anything abnormal. Sometimes, however, small areas of ecchymosis are found beyond the margins of the patch, especially in the direction of the pylorus.

The cuticular mucous surface appears, as a rule, quite normal. In a few instances a certain amount of desquamation has been noticed.

The "port wine patch" can only originate shortly before death.

The microscopical appearances of the port wine patch show that the condition is quite recent, and must have necessitated a profound interference with the functions of the stomach. The process of digestion does not, however, appear to be in any way impaired in the sick animal; it is the rule to find that the stomach is well filled with properly masticated, unfermented food, thus showing that, up to a short time before death, the animal was feeding in the usual way. The conclusion is inevitable that *the change in the stomach is brought about just before death.* The nature of the change will be referred to later, but it should be remarked here that the change, like that in the lungs and elsewhere, is not, in any sense, an inflammatory one.

Intestines.

The Intestines.—Out of 35 cases the intestines showed some abnormality in 16 instances, i.e., in about 45 per cent. The change usually took the form of petechial hæmorrhages in the duodenum or rectum, and purple blotches at the appex of the cæcum and throughout the colons. Punctate hæmorrhages, or large patches of apparent injection, were noticed in the jejunum and ileum in

4 instances. In only one case were the intestinal changes very marked, and consisted of intense injection of the mucous lining of the whole large gut from the ileo-cæcal valve to the anus.

THE SPLEEN rarely presents any visible abnormality. Its size and consistence are not altered, and the interior of the organ has the appearance seen in health. The weight, however, is nearly always distinctly in excess of the normal. The outer (diaphragmatic) surface may show numerous, minute, rounded, raised, dark red spots.* Spleen.

THE LYMPHATIC GLANDS appear, as a rule, quite normal.

THE LIVER.—Out of 35 cases the liver is noted as having some abnormality in eighteen. This abnormality has usually taken the form of an engorgement of the whole organ with blood. On three occasions the cut surface has revealed an appearance similar to that of an early stage of the "nutmeg" condition; subserous petechiæ have been noted twice. Liver

THE KIDNEYS.—Out of 29 instances in which the naked eye appearance of the kidneys has been recorded, it is stated to be normal in 6 only. The usual change observed has been a congestion, sometimes extreme, of the cortical, and an unnatural paleness of the medullary regions. Broad, dark striae have often been noticed running in the medullary rays. In only one case could the presence of changes (hæmorrhages), very obvious with the microscope, be detected by the unaided eye. Amber jelly was occasionally found beneath the adventitious capsule and around the pelvis of the organ. Kidneys

THE URINE.—A fair sample of the urine has been obtained from the bladder on seven occasions. The density of these samples has varied from 1,008 to 1,032; none contained blood or renal casts; three contained a mere trace of albumen, and in one albumen to the extent of one tenth was found. The reaction varied from faintly to strongly acid. Urine.

THE BLADDER, as already hinted, has usually been found empty of urine. It has, however, very often contained a few drachms of a viscid, puriform fluid of the same nature as that described on page 21, and which had evidently passed into it from the ureter. Bladder.

The vesical mucous membrane has, on several occasions, appeared to be mottled with light purple patches. This light purple mottling has also been observed in the lining of the UTERUS.

* It is possible that these dark red spots are to be found upon the surface of the spleen of the undiseased animal.

An in-foal mare had not aborted.

In the only instance observed of a pregnant mare dying of the disease the animal had not miscarried. The fœtus (about a seven-months), membranes, and uterus in this case appeared quite normal.

The interior of the long bones.

The Marrow of the Long Bones.—Sections of one or more of the long bones have been made in the last twenty one necropsies held at the Laboratory† and the following statement can be made:—

In every fatal case of the disease the marrow of both thigh bones has been found obviously abnormal, and in many the marrow of one or both humeri was also affected. All the remaining bones of the extremities have appeared normal.

Some animals which were believed to be immune from the disease and which had died from other causes showed a precisely similar but less developed change in the interior of the thigh bone.

The abnormal appearance in the interior of the thigh bone may be thus briefly described:—

The cancellous tissue of *the head and upper extremity* of the bone is much redder than that of the distal extremity.

The yellow marrow in the cavity of the shaft is infiltrated in its upper part, and usually more on one side than the other, by a soft, maroon material. The greatest thickness or development of this material is in the medullary cavity immediately adjacent to the entrance of the main nutrient artery. The appearances very strongly suggest the idea that the red marrow of the head of the bone has grown down through the yellow marrow and is escaping by way of the main nutrient vein. In no case has abnormality of the distal half of the bone been noticed.

The material which invades the yellow marrow is of a different consistence to that of its surroundings, being usually softer and less elastic to the touch. It is clearly defined by an abrupt margin from the yellow marrow in which it is embedded.*

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† Only six of these cases are included in the tabulated notes appended to this Report.

* Since writing the above, a necropsy has been made upon an animal aged 12 years, which revealed a still more remarkable abnormality in the marrow of the femur. The appearance in the head of the bone and the presence of the maroon material extending down to the nutrient foramen were the same as in other cases. The yellow marrow had, however, entirely disappeared, its place being taken by a clear, firm, amber-coloured jelly. This jelly was quite devoid of fat, and had a purely proteid composition. The subject was not emaciated.

The microscopical examination of this maroon "marrow" proves to be of exceptional interest, and may afford a clue not only to the pathology of the final stage, but also to that of the stage of incubation.

Appearances in the body which may mislead.

It is not out of place, at the conclusion of this account of the naked eye morbid anatomy of Horse-sickness, to refer to certain appearances which may be found in the body apart from this disease. Although frequently found in the body of the Horse-sickness animal it is probable that they should be classed among normal phenomena.

The Parietal Peritoneum covering the diaphragm, especially in its muscular portion, and that in relation with the outer surface of the spleen, is found to be overlaid with a fine lacework of soft, milk-white material. This material is readily detached by the finger-tip.

The surface of the Liver, especially the upper aspect of the right lobe, carries one or more large, circular areas, which are shaggy, with firmly attached, though delicate, tags of a tough, white material.

The mucous membrane of the Duodenum, and occasionally that also of the Jejunum, may be finely stippled with pigment of a steel-grey colour.

The Pelvis of the Kidney and the lumen of the Ureter are often found to be filled with a creamy, puriform fluid. If this material be not present in the renal pelvis it can nearly always be made to exude from the papillæ of the gland by gentle pressure. As the fluid has all the appearance of pus, its discovery might readily mislead the inexperienced pathologist. The fluid is present in especial abundance if the necropsy has been delayed; it does not appear to be present immediately after death. Microscopically the fluid is found to consist of innumerable epithelial cells, which have the appearance of being derived from the convoluted tubules of the kidney.

MICROSCOPIC APPEARANCES.

The Marrow of the Long Bones.—Sections made through the junction of the maroon material and the normal yellow marrow show that the unusual appearance is due (1) to an engorgement of the capillaries and veins of

the marrow with blood, and (2) to an infiltration of the intercellular spaces with a multitude of new elements.

The fat cells, which form the great bulk of yellow marow, are not altered in appearance.* The position of the small amount of lymphoid tissue usually found among these cells is taken by large capillary-walled sinuses and masses of cellular elements. In many fields the sinus-like capillaries appear to possess incomplete walls, and the blood with which they are distended thus seems to be in direct contact with the masses of cells. Some of these cells appear among the blood corpuscles in the sinus, and some blood corpuscles appear among the cells of the cell-masses.

The elements of the marrow-infiltration.

The elements composing the cell-masses, or rather the cell-infiltration, for the aggregations of cells are continued into one another, may be briefiy described as follows:—

1. By far the most conspicuous elements are lumps of a golden-brown material. These objects are of irregular shape, and of a diameter which varies from 3μ to 26μ. They do not stain with carmine, methyl blue, haematoxylin or eosin, and have therefore the same appearance in the stained as in the unstained preparation. The typical brown lump is composed of rounded nodules closely applied to one another thus giving somewhat the appearance of an early segmenting ovum. The larger lumps do not show an increase in the number, but rather of the size of their component nodules. Some of the nodules of the larger brown lumps appear broken up into smaller fragments and collections of these fragments may be seen at some distance from the parent mass.

   The material of these brown, modulated masses contains much iron for it becomes of a deep blue colour when treated with ferrocyanide and hydrochloric acid.

2. Marrow Cells (Myelocytes). These appear as large and somewhat angular cells with a diameter varying from $6{\cdot}5\mu$ to 15μ (usually 13μ). The nucleus is relatively very large (7μ) and is usually of oval outline, but sometimes lobed or reniform; it is usually situ-

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* See foot-note on p. 20.

ated at the periphery. The nucleus stains deeply with gentian violet, but the body, which is nearly hyaline, is with difficulty stained by any reagent. The body of the cell is often found reduced to a mere shred attached to one side of the nucleus.

The large, oval nucleus of these cells often show a clear, central vacuole around the margin of which are arranged a number of dark particles.

3. Spherical or oval bodies, 7μ to 14μ in diameter, which appear to be the enlarged nuclei of the elements of Class 2 deprived of all envelope. They stain but feebly with nuclear stains and often show a clear, central vacuole and dark granules scattered throughout their substance.
4. Erythroblasts and Small Lymphocytes.
5. Giant Cells (Myeloplaxes). In addition to the typical myeloplax, deeply-staining, irregular lumps of protoplasm, 12μ to 17μ in diameter, and without any distinguishable envelope are occasionally found.
6. Large cells, 10μ to 14μ, with a comparatively small, single nucleus placed peripherally. The body of the cell is coarsely granular but the granules are not eosinophile.

The infiltrated marrow in non-fatal cases of the disease differs from that in the fatal only in the absence from it of the elements described under Class 1.

The maroon material, the elements composing which have been thus described, differs but slightly in its components from normal red marrow, the most important variation being the presence of the golden-brown nodules. It is possible that the large elements described in Class 6 are also strangers to the normal red marrow.

The mode of production of the masses of golden-brown material (Class 1) is not quite certain, they appear, however, to originate by a species of degeneration of the elements of Classes 2 and 3. The nucleus in Class 2 increases in size until little or no envelope remains; the whole cell becomes of a yellowish colour; black, pigmented particles appear in the nucleus which takes on a golden-brown tint and begins to divide up into rounded segments; some of the resulting nodules are seen to carry specks of black pigment.

Whether the cells which undergo this remarkable degeneration are themselves of a parasitic nature (having merely the appearance of myelocytes) or whether the parasite of the disease invades the myelocytes and induces this degeneration are suppositions to be dealt with in future research.

The Lungs.—Whatever be the naked eye appearance of the lungs (congested, hæmorrhagic, œdematous, or marked by tracts of amber jelly), the one essential factor of the disease in its final stage can readily be discovered in all.

The smaller vessels of the pulmonary system, arterioles, capillaries, and venules, will be found to contain not only normal blood corpuscles, but also many "foreign" cells similar to those found in the bone-marrow. In the larger vessels these cells may appear either discrete or aggregated into clumps. There appears to be a tendency for the cells to remain separate in the arterioles and to be aggregated in the venules. About one third of the cross-sectional area of a small artery or vein is usually taken up by these new elements. When appearing in any numbers the cells are found to be in contact with the vessel wall and not lying in the "fair way" of the vessel. The capillaries in the alveolar septa are often seen to be occupied by a single chain of these "foreign" cells.

The new elements thus occurring in the pulmonary vessels appear to be identical with those described, under classes 2, 3, 4, and 6, in the marrow of the long bones. The diameter of cells of Class 2 is often found to be as much as 17 μ. Among the vessel-contents a roughly quadrilateral mass of yellowish, hyaline material is occasionally visible (?Class 1 of marrow elements).

A very rare variety of "cell" within the pulmonary vessels may be described here because it has also been found in the stomach capillaries and in the bone-marrow. Its diameter is from 22μ to 26μ. A delicate cell-wall seems to be present, and encloses a clear, hyaline material in the centre of which lies a yellowish, granular mass of about 12μ in diameter. Within the central mass is yet another body, which appears as a deeply-stained nucleolus.

The erythrocytes among which the foreign cells lie appear quite normal, and have a diameter of from 3·75μ to 5μ; the leucocytes present measure from 4μ to 8μ.

The gelatinous, subpleural transudate appears structureless. The meshes of the fibrous basis of the pleura are distended with the material, and the serous endothelium thus appears to be carried away from the lung.

The alveoli are often seen to be flooded with transudate, which thus gives to the section an appearance of having been embedded in some homogeneous matrix. The alveoli, although filled and surrounded by the transudate, retain their normal shape and have never been found collapsed. There does not appear any trace of small-celled infiltration either of the conective tissue framework or of the alveolar contents. The transudate in the air cells frequently contains one or two "foreign" cells.

The lumen of the bronchioles is usually more or less obstructed by a hyaline material of similar appearance to the transudate in the alveoli. The periphery of this content has the appearance of being built up of a number of small, spherical masses of the material.

The connective tissue framework and the alveoli may often be found flooded with blood instead of with transudate. In these specimens the blood within the alveoli contains many of the "foreign" cells described as occurring in the pulmonary vessels.

THE HEART.—Sections of the heart made at the site of the subserous ecchymoses in its interior reveal the following changes:—Some of the capillaries in the myocardium and many in the loose fibrous tissue of the endocardium are distended with blood. Aggregations of cells, resembling those of classes 2, 3, 4, and 6 in the maroon marrow, can be found plugging some of the smaller arterioles. Immediately beneath the endothelium and among the subjacent layers of elastic and dense fibrous tissue, planes of extravasated blood appear. The corpuscular elements in these extravasations are seen to occupy all the interstices of the tissue; beneath the endothelium they are so closely packed that all other structures appear obliterated.

Description of the normal mucous membrane of the villous stomach.

THE STOMACH.—The normal glandular layer of the mocous membrane of the stomach, in the region in which the "port wine patch" occurs, consists of a multitude of closely-packed, parallel tubular glands. These tubes run a straight course from their bases, which rest upon a muscularis mucosæ, with but a trace of intervening lymphoid tissue, to their openings upon the surface of the stomach. The upper portions (ducts) of all the tubules are often seen to be inclined towards one side; the glands then open obliquely to the surface and not at right angles. Among the narrowed bases of the glands, where they rest upon the muscularis mucosæ, triangular spaces are seen. These triangular spaces are

occupied by lymphoid tissue and by the anastomosing branches of a capillary network around the bases of the gland tubules. From the upper side of this anastomosis capillaries arise which pass upwards among the tubules to the surface. Arrived near the mouths of the glands these capillaries again form an anastomosis and from the lower side of this, "return capillaries" descend among the tubules to penetrate the muscularis mucosæ and join the veins in the submucous layer.

Below the submucous layer lie the muscular coats of the stomach: these, however, have no particular interest for the present investigation. Sections made through the "port wine patch" show that all abnormalities are confined to the mucous and submucous coats.

Abnormal contents of vessels in submucosa.

Submucous Layer.—Many of the small arterial branches passing through the submucous areolar tissue to enter the glandular layer are seen to contain masses of cells having the same appearance as the masses of "foreign" cells described as occurring in the pulmonary vessels. A typical cell-mass in a submucous arteriole measured 93μ by 25μ.

The component elements of these cell-masses may be stated, for the sake of brevity, to be identical in all characteristics with those of Classes 2, 3, 4, and 6, in the bone-marrow.

In company with the preceding elements a large (18μ to 20μ) irregular fragment of unstained, semi-crystalline, yellowish material is occasionally seen.

When the contents of the veins are compared with those of the arteries it is seen that the foreign cells are very much less numerous in or quite absent from the former.

Small extravasations of blood are occasionally seen in the submucous layer, but it is very seldom that any indications of the presence of a transudate occur.

Abnormalities of the mucosa.

The Glandular Layer.—This layer presents a very striking appearance even under a low power. Many of the capillaries which lie between the gland-tubules will be seen to be enormously distended with blood. The diameter of the distended capillary may be as much as 100μ. The "port wine patch" is due solely to the presence of these engorged vessels.

The muscularis mocosæ, the interglandular lymphoid tissue, and the elements of the glands themselves do not show any recognisable abnormality.

Many normal capillaries, without contents, may be seen between the tubules; it is only here and there in the field that distended capillaries are seen.

The engorged capillary is usually found to reach much nearer to the surface than to the base of the mucous membrane. The vessel appears to contain normal blood, being tightly packed with erythrocytes and a few leucocytes. Elements of the "foreign" cell-masses are, however, occasionally seen.

Among the bases of the gland-tubules sinus-like dilations of capillaries appear. In most of these dilations masses of "foreign" cells, rounded granular fragments and half crystalline plates are plainly seen.

Near the surface of the mucous membrane extravasations of blood, due to rupture of the engorged capillaries, are frequently found. The margin of the section may show a fraying away of the glandular tissue, due apparently to necrosis from deficient blood supply.

The Spleen.—Numerous, iron-containing, nodular masses, similar to those described under Class 1 in the marrow, are found in abundance among the cells of the spleen-pulp. Elements corresponding with those of Classes 2, 3, 4, and 6 in the marrow are also usually to be found with ease; the bulk of the section, however, consist of normal spleen-pulp. Sections of the spleen occasionally reveal masses of crystals of a reddish-orange hue. These crystals are arranged for the most part in bundles resembling a loosely-tied wheatsheaf, and recall those of tyrosin. They do not give the iron reaction and do not stain with ordinary stains.

The Kidney.—In every section it is, as a rule, possible to discover a collection of "foreign" cells and semi-crystalline fragments impacted in the small afferent vessel of a glomerulus. Engorgement of capillaries and interstitial haemorrhages are, however, area for area, much less numerous than in the stomach. This fact is probably accounted for by the more completely terminal character of the renal circulation.

The interstitial and glandular tissues of the kidney do not present any other abnormality.

The Rectum.—Microscopic appearances of essentially the same changes as those found in the stomach may often be discovered in the mucous membrane of the rectum.

CONCLUSIONS.

The briefest consideration of the microscopic appearances in the various affected organs justifies the conclusion that all the lesions of the "stage of death" are the results of CAPILLARY EMBOLISM, the term "stage of embolism" may therefore now be substituted for that of "stage of death."

The emboli consist of certain cells and other bodies which are present in great abundance in the diseased marrow; that they are originally confined to this situation, and only enter the circulation at the commencement of the final stage of the disease is very probable but not yet conclusively proved.

The channel by which the emboli gain entrance to the veins, whether directly by the efferent nutrient blood vessel or by the more difficult and less direct route of the lymphatics and thoracic duct, has yet to be ascertained.

The results of the sudden blocking of an arteriole comprise:—

The changes which usually follow the sudden blocking of a small artery.

1. A temporary local anæmia.
2. A reflux engorgement of its capillaries with blood from anastomosing neighbours.
3. A transudation of fluid, if the surroundings permit, from the engorged capillaries.
4. A giving way of the walls of some of the engorged capillaries and the formation of small ecchymoses.

When the fact is recognised that localised capillary engorgement, the transudation of coagulable or non-coagulable fluids, and hæmorrhagic extravasations are all phenomena of the same process, it is obvious that only one explanation is necessary to account for the distribution throughout the body of all or any of the morbid changes found in fatal Horse-sickness. The only change, the position of which must be explained by itself, is that which is found in the marrow of the long bones. This change, as already stated, may prove to be the fountain head of the disease.

For the sake of brevity and clearness a dogmatic statement will now be made of the course of events in the final stage of the disease:—

A "dogmatic" statement of the events of the final stage of the disease.

At the end of the period of fever, "cell-masses" become liberated from the diseased bone-marrow. Having gained the large veins, these masses are carried to the right side of the heart and from thence to the lungs: here they become impacted in the smaller branches and capillaries of the pulmonary artery.

One of two things may now happen:—

1. If the area of breathing tissue, thus suddenly put out of action, be sufficiently large, the animal is rapidly asphyxiated. In such a case, beyond the marrow-change, a small collection of *fluid of very low density in the pleural sacs* and a scarlet colour in the interior of the lungs, no morbid change whatever will be discovered.

2. In the great majority of cases the animal survives the embolism of its pulmonary capillaries for several hours or even days. The fluid transudate in the pleural sacs, which is being derived from the pulmonary venules, rapidly increases and further events ensue.

As the result of the breaking up of the impacted cell-masses into smaller groups of individuals, and owing to the great dilatability of the pulmonary capillaries, emboli now begin to escape into the pulmonary veins. Arrived in the left ventricle the "foreign" cells are driven into the aorta.

It has been already remarked, p. 24, that the cell-masses tend to lie against the vessel-wall and not to occupy the centre of its lumen. A result of this peculiarity is that some of the first instalment of embolic masses nearly always find their way into the nearest lateral branches of the great vessel. We have in this an explanation of the great frequency with which dropsy of the pericardial sac and subendocardial ecchymoses occur, for the first lateral branches of the aorta are the Cardiac (or Coronary) arteries.

The pericardial transudate is œdema-fluid which has been expressed from the sponge-work of the heart-muscle by its continuously repeated contractions.

The subserous ecchymoses represent blood which has been extravasated in the heart substance (as a consequence of the giving way of engorged capillaries) and which, like the transuded fluid, is squeezed out in the direction of least resistance.

Next to the coronary branches comes the great Anterior Aorta. Emboli enter this also, but, owing to the regions supplied, the effects of embolism are not grossly apparent. The lodgment of emboli in the cerebral cortex produces the sudden staggering or falling to the ground which is sometimes observed, and may, of course, result in sudden death.

Next to the Anterior Aorta comes the Broncho-œsophageal trunk supplying the posterior end of the œsophagus, the bronchial glands and tissues a-

round the roots of the lungs, and *all the connective tissue of the lungs themselves with their coverings.* Embolisation of the branches of this artery results in a transudation around the roots, in the connective tissue and beneath the pleura of the lungs. The anterior mediastinum will also be affected.

The rapid reflux of blood into the blocked capillaries produces, with especial ease, a transudation into the distensible framework of the lungs. This transudate is at first necessarily restricted in its distribution to the areas supplied by the blocked arterioles. It is in this way that localised subpleural and interlobular deposits of amber jelly and subpleural and interstitial hæmorrhages are produced.

A further important result of the embolisation of the bronchial vessels is that the liquid transudate which has already escaped into the pleural sacs (in consequence of the antecedent obstruction of the pulmonary capillaries) now receives large accessions. The transudate derived from the bronchial vessels is probably of higher specific gravity than that from the pulmonary capillaries.

Next to the Broncho-œsophageal vessel comes the Cœliac Axis. The divisions of this vessel are the Hepatic, the Splenic and the Gastric. The Hepatic artery is small and its distribution unfavourable to the development of obvious transudations. Most of the radicles of the Splenic artery open directly into the meshes of the spleen-pulp, into which the emboli are precipitated without any obvious effects. The Gastric artery supplies, by its two divisions, the villous half of the stomach. This vessel is particularly well fitted to demonstrate the effects of embolism in its capillaries. The terminals of the artery are the capillaries lying between the gastric gland tubules, and they are often separated from the surface by a single layer only of translucent cells. Reflux engorgement of the venous capillaries in the villous mucous membrane is therefore especially apparent, and results in the production of "the port wine patch."

The cuticular portion of the stomach is lined with a thick layer of stratified epithelium resembling the skin: capillary engorgement in the areolar coat of this portion would not, therefore, be apparent to the unaided eye.

The apparent selectiveness exhibited by the villous half of the stomach appears in this way to be merely the result of anatomical peculiarities.

If the parietal branches of the Posterior Aorta be embolised a transudate

(amber jelly) will escape into the regions supplied by the Intercostal, Diaphragmatic and Lumbar arteries.

An explanation of why the amounts of the pleural and pericadial transudates often appear in inverse ratio can now be suggested. If the emboli be entirely arrested for many hours in the capillaries of the lungs the resulting pleural transudate will be excessive in quantity; there will be little or no pericardial transudate, because the cardiac vessels are, as yet, unaffected. If, on the other hand, the emboli traverse the lung capillaries more rapidly the pleural transudate will be small in quantity; the cardiac capillaries becoming speedily embolised, the pericardial transudate will be excessive.

The whole statement may be summarised thus:—

Death in Horse-sickness is due to capillary embolism or its results.

The emboli consist of certain cells and masses of semi-crystalline, pigmented material.

The medullary cavities of the thigh bones, and also sometimes of the arm bones, contain these cells and pigmented material in great abundance.

The ultimate distribution of these emboli is, to some extent, accidental; in the main it is dependent, as are also the obvious results of the embolism, upon general anatomical considerations.

The explanation of why the various clinical varieties of the disease, "Dikkop," "Pericardial," "Pneumonic," "Intestinal," etc., verge into one another in so confusing a manner is now apparent. The uselessness of these terms, except in a strictly clinical sense, is still more apparent.

In connection with overgrowth of red-marrow, which, as already stated, is probably the primary lesion of the disease, the following physiological observation is of interest. When a sudden demand arises for a large supply of red corpuscles, as is the case, for example, after an excessive loss of blood, the red-marrow in the heads of the long bones rapidly hypertrophies and spreads down into the cavity of the shaft. In Horse-sickness we find a similar hypertrophy of red-marrow occurring, but, as far as we can see, without any special need for it; not only is the hypertrophy uncalled for but it causes the death of the animal.

If a blood-count be taken just before death the leucocytes will be found to be moderately increased in number and the red corpuscles to be about double the number which they were the day previously. These changes in the blood-count are suggestive, although a possible explanation is to be found in the diminution of the plasma owing to the formation of the transudates.

The correctness of the scheme of events as given above requires verification by other observers. If the conclusions be confirmed, the disease will be shown to belong to one of an entirely novel class, to which some such name as "Critical Myelæmia" or "Myelocytic Embolism" might appropriately be given.

The scheme has been termed a "dogmatic statement," not because of any doubt as to the accuracy of any one observation, but from a feeling of diffidence towards the opinions of the many previous workers in the pathology of the disease.

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January 24, 1904.

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SYNOPTIC TABLE of Morbid Appearances found in 35 Cases of FATAL HORSE-SICKNESS.

Number of Subject.	Time since Death (in hours).	Side on which Body has lain since Death.	Foam-cloud.	Coarseness of Muzzle.	Swelling of Temporal Fossæ.	Amber Jelly in Peripheral Parts	Amber Jelly in Jugular Channels	Total quantity of Pleural Transudate. (in c.c.)	Quantity of Transudate in Right Pleural Sac.	Quantity of Transudate in Left Pleural Sac.	Density of Transudate in Right Pleural Sac.	Density of Transudate in Left Pleural Sac.	Appearance of Pleural Transudate.	Weight of Lungs (in Kilogrammes)	Condition of Visceral Pleura.	Condition of Lungs.	Amber Jelly at Roots of Lungs.	Quantity of Pericardial Transudate (in c.c.)	Density of Pericardial Transudate.	Appearance of Pericardial Transudate.	Amber Jelly among Vessels at Base of Heart.	Appearance of Epicardium.	Appearance of Endocardium	Port Wine Patch in Stomach.	Intestines showing Vascular change.	Weight of Spleen (in Kilogrammes)	Weight of Liver (in Kilogrammes)	Weights of Kidneys (in Kilogrammes) R.	Weights of Kidneys (in Kilogrammes) L.	Weight of Heart (in Kilogrammes)	Marrow of Long Bones.	Remarks.
80	½	R	—	—	—	†	—	250	225	25	.	52	Blood-stained	12·247	H	Congested	—	200	16	Turbid, Red	†	Normal	Ecch. (L Vent.)	Normal	Duod. Cæc. Colon	1·701	11·566	1·474	1·360	.	.	Large Animal. Two Crops of Sub-pleural Petechiæ of different ages.
78	½	R	—	†	†	†	† (Slight)	375	225	150	14	14	Turbid, Orange	9·298	N	Congested	—	975	16	Pale Straw Coloured	†	Pet. J	Ecch. (R & L Vent. / L Aur.)	N	N	1·134	5·329	·453	·510	3·62	.	Amber Jelly beneath Pleura covering Diaphragm.
92	½	R	—	—	—	—	—	1,450	1,150	300	17	18	Straw Coloured	11·340	H J	H J	—	50	.	Reddish Amber	—	Pet. J	Ecch. (R & L Vent.)	Slight	N	2·041	5·599	·623	·567	3·4	.	
60	1	.	†	†	—	—	—	6,000	3,000	3,000	.	18	Straw Coloured	14·061	J	Congested	†	250	16	Pale Straw	—	N	Ecch. (L Vent.)	Moderate	Duod.	.	6·804	.	·907	4·3	.	Large Animal.
47	1	L	† (Slight)	†	—	—	—	3,350	1,600	1,750	.	19	Straw Coloured	8·845	H J	Congested, Œ	—	30	.	Pale Straw	†	J	Ecch. (L Vent.)	Slt.	Duod. Cæcum	·822	4·252	·453	·453	2·6	.	Small Animal.
81	1	.	†	—	—	—	—	290	210	80	12	15	Amber Coloured	16·102	H J	H Œ J	—	90	14	Pale Straw	†	J	.	Slt.	N	1·304	7·484	·793	·680	3·14	.	
84	1½	R	—	—	†	†	† (Slight)	1,325	1,050	275	16	18	Turbid, Reddish	18·597	H J	H J Œ	†	25	.	Pale Straw	—	N	Ecch. (L Vent.)	V Slt.	Ileum	1·644	7·144	·737	·793	3·62	—	Large Animal. Humens only bone examined
50	2	.	—	—	†	† (Slight)	†	60	30	30	.	.	Turbid, Reddish	5·443	N	Congested	—	200	17	Pale Straw	—	N	Ecch. (L Vent.)	V Slt.	All large Intestine	1·020	4·989	·510	·510	2·94	.	Small Animal.
51	2	R	—	—	†	—	—	4,300	3,500	800	16	.	Straw Coloured	11·907	J	J (Slight)	†	550	15	Pale Straw	†	J	Ecch. (L Vent.)	Mod.	N	1·247	6·350	·850	·680	4·76	.	Large Animal.
C	2	.	—	†	—	†	† (Slight)	113	70	43	19	20	Reddish Amber	6·350	J (Slight)	J (Slight)	†	597	18	Pale Straw	—	Pet. J	Ecch. (L Vent.)	V Slt.	N	1·587	8·958	·907	·907	3·17	.	
48	2	R	—	—	—	.	.	1,210	750	460	20	20	Straw Coloured	13·041	J	Œ	†	400	17	Pale Straw	—	N	Ecch. (R & L Vent.)	Slt.	Duod.	1·304	5·896	·567	·567	3·85	.	Small Animal.
64	3	R	†	†	—	—	—	4,450	3,000	1,450	.	15	Straw Coloured	9·695	J	H Œ J	†	75	14	Blood-Stained	—	N	Ecch. (R & L Vent.)	Severe	Duod.	1·474	4·762	·652	·623	3·17	.	Petechiæ beneath mucous membrane of Gums and Lips.
76	3	L	—	†	—	† (Slight)	—	170	100	70	14	16	Amber Coloured	15·876	H J	J	—	900	16	Straw Coloured	—	J	Ecch. (L Vent.)	Slt.	N	1·360	7·938	·680	·680	3·88	.	Large Animal.
36	4	.	†	—	—	—	—	4,000	2,000	2,000	.	18	Straw Coloured	7·267	J	Œ J	†	15	.	Straw Coloured	—	N	N	Mod.	Cæcum.	1·020	5·443	.	.	4·08	.	Pregnant. Had not aborted.
99	4	R	—	†	†	† (Slight)	†	350	150	200	16	17	Red-orange	9·355	H	N	—	1,380	16	Straw Coloured	†	Pet. J	Ecch. (R & L Vent.)	Sev.	N	1·644	6·690	·623	·567	4·08	.	
63	5	R	†	—	—	† (Slight)	†	1,400	1,080	1,220	20	20	Straw Coloured	11·283	J	H	†	50	.	Straw Coloured	†	N	Ecch. (L Vent.)	Mod.	N	1·020	5·840	·680	·680	2.09	.	
40	5½	.	—	†	†	†	†	700	500	200	.	.	Straw Coloured	.	H	H	—	500	.	Straw Coloured	—	Pet.	Ecch. (R & L Vent.)	Sev.	Duod. Rectum	1·162	5·273	·623	·595	.	.	Small Animal.
101	6	R	—	—	—	—	—	490	290	200	28	24	Dirty Red	10·092	H	Congested	—	10	.	Light Orange	—	Pet. J	Ecch. (R & L Vent.)	Mod.	Ileum and all large Intestine	2·097	6·350	·793	·793	3·68	.	
58	6½	L	—	—	—	—	—	0	.	.	.	.	Dirty Red	8·505	N	Congested	† (Slight)	1,200	19	Amber Coloured	†	J (Slight)	N	Mod.	Colon	2·835	7·824	·765	·793	6·97	.	
27	8	L	—	—	†	† (Slight)	† (Slight)	300	100	200	.	.	Blood-stained	4·621	N	Congested	—	325	.	Straw Coloured	—	N	Ecch. (R & L Vent.)	Slt.	Cæcum	1·134	4·932	·481	·453	2·49	.	Small Animal.
44	8	L	†	—	—	—	—	4,700	2,700	2,000	22	.	Cherry Red	8·618	J	Congested	†	250	18	Dark Cherry	—	N	Ecch. (R & L Vent.)	Slt.	N	·850	4·762	.737	·538	.	.	
75	10	R	—	†	†	† (Slight)	† (Slight)	1,120	575	550	22	22	Port Wine	8·752	H	H	—	1,200	16	Brown Sherry	—	N	Ecch. (L Vent.)	Mod.	N	1·020	4·989	·694	·652	2·69	.	Mesentery of small intestine injected.
115	10	R	—	†	†	—	†	225	125	100	21	21	Red Amber	9·185	N	Congested	† (Slight)	770	17	Amber Coloured	—	Pet.	Ecch. (L Vent.)	Mod.	N	1·701	6·804	·648	·765	4·08	†	
114	10	.	—	†	† (PM)	† (Slight)	†	580	250	330	22	22	Clear, Reddish	5·896	H	Cong. (Slight)	—	970	16	Straw Coloured	—	Pet.	Ecch. (R & L Vent.)	Sev.	Rectum	1·984	6·350	·737	·680	4·49	†	Mesorectum injected.
66	10	.	—	—	—	† (Slight)	—	300	130	170	20	20	Clear Light Red	4·762	H	Œ	—	175	18	Straw Coloured	—	N	Ecch. (R & L Vent.)	Slt.	Duod. Rectum	1·304	4·309	·567	·510	2·26	†	
83	12	.	—	—	—	—	—	20	10	10	.	.	Clear Light Red	8·618	.	H Œ Cong. J	† (Slight)	0	.	.	—	N	Ecch. (L Vent.)	Slt.	Rectum	1·587	5·329	·737	·680	2·6	†	Examined immediately after, and again twelve hours later.
43	15	R	—	—	†	† (Slight)	.	500	250	250	.	24	Clear Amber	8·391	J	J (Slight)	†	200	22	Dark Straw Coloured	—	.	.	Slt.	N	2·948	5·443	·850	·648	2·97	.	
61	18	R	—	—	—	—	—	670	430	240	30	28	Blood Coloured	6·577	N	N	—	500	25	Claret Coloured	—	N	Ecch. (L Vent.)	Sev.	N	1·360	4·564	·850	·737	4·3	.	
79	18	L	†	—	—	—	—	5,100	2,350	2,750	19	19	Blood-stained	9·582	J (Slight)	J (Slight) Œ	—	550	16	Port Wine Coloured	—	N	Ecch. (L Vent.)	Slt.	N	·850	4·280	·737	·850	2·6	.	
87	18	R	Fluid	† (Slight)	—	—	—	5,520	2,740	2,780	22	22	Cherry Red	11·113	J	J Œ	†	150	25	Dark Red	—	N	Ecch. (L Vent.)	Slt.	Cæcum Colon	2·664	6·123	·878	·963	3·68	†	
93	18	L	†	†	†	—	—	5,500	.	.	.	20	Cherry Red	8·164	J	H	—	150	16	Dark Purplish Red	†	N	N	N	N	1·530	5·273	·680	·680	2.49	.	
49	20	L	†	†	†	†	†	5,650	2,750	2,900	18	18	Orange Red	5·783	J	N	—	100	18	Dark Red	—	N	P M Staining	Mod.	N	1·757	4·791	.	.	2·26	.	
42	21	R	—	—	—	—	—	2,500	1,250	1,250	.	.	Dark	9·072	N	N	—	550	18	Dark Red	—	N	P M Staining	Mod.	N	1·247	4·195	·510	·510	3·57	.	
59	22	R	—	—	†	†	†	1,000	.	.	.	.	Like Blood	11·340	J	H	Hæmorrhagic Fluid	100	22	Blood Coloured	—	N	P M Staining	Sev.	N	1.814	5·216	·680	·680	4·42	.	
85	22	R	†	—	—	—	—	1,450	700	750	32	26	Clear, Dark	13·608	J	Congested	—	600	26	Reddish Brown	—	N	Ecch. (L Vent.)	Mod.	N	1·134	5·443	·765	·680	3·11	.	Large Animal.

ABBREVIATIONS USED.—† Present. — Absent. . Not recorded. P M Developed after Death. R Right. L Left. N Normal. V Slt. Very Slight. Slt. Slight. Mod. Moderate. Sev. Severe. H Hæmorrhages. J Amber Jelly. Œ Œdema. Pet. Petechiated. Ecch. Ecchymosed. Cong. Congested. Duod. Duodenum. Cæc. Cæcum. Vent. Ventricle. Aur. Auricle.

NORMAL WEIGHTS OF CERTAIN ORGANS IN KILOGRAMMES:—Heart...3·06, Liver...4·88, Spleen...·9, Right Kidney...·76, Left Kidney...·7, Lungs...5·89.

NOTE.—Cases are sometimes referred to in the text, the details of which are not included in this table.

found in 85 Cases of FATAL HORSE-SICKNESS.